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RULE 26 EXPERT REPORT OF
PATRICIA G. MOORMAN, MSPH, PHD

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Patricia G. Moorman

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Scientific Review of the Epidemiologic Evidence on Talc Use and Ovarian Cancer

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Background and Qualifications of Patricia G. Moorman, MSPH, PhD

I am a tenured professor in the Department of Community and Family Medicine, Duke University School of Medicine, Durham, NC and a member of the Cancer Control and Population Sciences Program in the Duke Cancer Institute. I am an epidemiologist with more than 25 years of experience in conducting research on women's health issues including ovarian cancer, breast cancer and menopause. Attached as Exhibit A to this report is a current copy of my curriculum vitae.

Education

I received a Bachelor of Science degree with distinction in pharmacy from the University of Kansas in 1980. I pursued graduate studies in epidemiology in the School of Public Health at the University of North Carolina-Chapel Hill, earning a Master of Science in Public Health (MSPH) in 1989 and a Doctor of Philosophy (PhD) degree in 1993.

Professional Experience

I have held positions in academic institutions since I completed my PhD, beginning as a research assistant professor in the Department of Epidemiology at the University of North Carolina-Chapel Hill from 1994 through 1996. From 1997 to 2000, I was an associate research scientist in the Chronic Disease Epidemiology division of the Yale University School of Public Health. I came to Duke University School of Medicine as an assistant professor in 2000, progressing through the academic ranks from associate professor, associate professor with tenure to my current position as professor in Community and Family Medicine. I also serve as the director of the Clinical Research Unit for the Department of Community Medicine and am a member of the Senior Faculty Advisory Committee for the Office for Research Mentoring in the School of Medicine. In addition, I am an adjunct faculty member in the Department of Epidemiology at the University of North Carolina-Chapel Hill.

Compensation and Testimony

My hourly billing is $400. I have given deposition testimony in one case (Gail Ingham, et al., v. Johnson & Johnson, et al., Case No. 1522-CC10417-01, Circuit Court of the City of St. Louis, Division 10) and have not testified at trial in the last four years.

Research Interests and Experience

My primary research interests are in the area of women's health issues, with a particular focus on studying racial differences in risk factors and outcomes. I have had funding from the National Institutes of Health (NIH) for more than 20 years, which has supported my research in ovarian cancer, breast cancer and ovarian function after hysterectomy. Three of the key studies in my research career are: 1) the African American Cancer Epidemiology Study (AACES), a multi-center, case-control study of ovarian cancer in African American women,[1] 2) the Carolina Breast Cancer Study, which is one of the largest studies focused on understanding racial differences in breast cancer risk and outcomes,[2] and 3) the Prospective Research on Ovarian Function (PROOF) Study, a cohort study designed to examine risk for ovarian failure after premenopausal hysterectomy.[3]

Each of these studies involved primary data collection, meaning that the investigative team designed the data collection procedures, developed the surveys, recruited study participants and obtained questionnaire data and biological specimens from the participating women. Each study has made unique contributions to the scientific literature.

AACES has enrolled more than four times as many African-American women with ovarian cancer than any other study and is providing the most comprehensive epidemiologic data on ovarian cancer risk factors in this population to date.[4-6] The Carolina Breast Cancer Study likewise provided key data on risk factors in African American women and was the first study to describe the markedly higher prevalence of the poor-prognosis basal subtype of breast cancer in young African American women.[7-11] The PROOF study is the largest prospective study of ovarian function after pre-menopausal hysterectomy and demonstrated that women

undergoing hysterectomy with ovarian conservation were at significantly increased risk for earlier menopause as compared to women who did not have a hysterectomy.[3,12]

Our study team published an analysis of talc exposure and ovarian cancer in 2016, using data from AACES. [13] This peer-reviewed paper, published in *Cancer Epidemiology, Biomarkers and Prevention*, was the first epidemiologic study of talc use and ovarian cancer that was focused exclusively on African American women. Our analyses found both a high prevalence of talc use in this study population and a statistically significantly increased risk for ovarian cancer among talc users. This paper was published prior to my involvement in litigation related to talc and ovarian cancer.

I have also been a co-investigator on the North Carolina Ovarian Cancer Study, which was a precursor to the AACES study. Data from this study were included in Terry, et al.'s [14] 2013 analysis of genital powder use and ovarian cancer that pooled from data from eight case-control studies. I am currently an investigator in the Ovarian Cancer in Women of African Ancestry (OCWAA) consortium. The OCWAA consortium, which was initiated in 2016, is a multi-center collaboration that aims to bring together data from case-control and cohort studies to evaluate similarities and differences between African American and white women in ovarian cancer risk factors and outcomes.

In addition to these studies, I am an investigator with the Evidence Synthesis Group in the Duke Clinical Research Institute, a team of researchers that conducts evidence reviews of the scientific literature. I have worked with this group on a number of systematic reviews and meta-analyses on women's health issues including an evaluation of the benefits and risks of oral contraceptive use for primary prevention of ovarian cancer [15-17] funded by the Agency for Healthcare Research Quality, and an evaluation of the benefits and harms of breast cancer screening[18] funded by the American Cancer Society to help inform their screening mammography recommendations.[19]

I am an author on more than 130 scientific publications, with more than 50 of them directly related to ovarian cancer. The ovarian cancer papers address a wide variety of risk factors including reproductive and hormonal factors, lifestyle characteristics, genetic factors, and talcum powder products. The main focus of the manuscripts on which I have been the lead

author has been ovarian cancer risk factors in African American women and the effects of reproductive characteristics, hormones and other medications on risk for ovarian cancer.[5,17,20-23] The papers have been published in some of the leading journals in the field of epidemiology, gynecology and cancer including the *American Journal of Epidemiology, Cancer Epidemiology Biomarkers and Prevention, Obstetrics & Gynecology* and *Journal of Clinical Oncology*.

My teaching experience includes courses in Cancer Epidemiology for graduate students in public health and Evidence-Based Medicine for physician assistant students. A primary emphasis of these courses has been for the students to gain an understanding of the advantages and disadvantages of different types of studies used in clinical and epidemiologic research. In particular, the Evidence-Based Medicine course is designed to help the students learn how to critically appraise the medical literature and apply findings to clinical practice. In addition, I have mentored at the individual level public health graduate students and medical students.

I serve as an editorial reviewer for numerous journals and have served as a peer reviewer of grant applications on several dozen study sections over that past twenty years. I have reviewed NIH grants for a variety of funding mechanisms ranging from small grants (R03) to large multi-project applications (SPORE grants and Program Projects). I also have served as both peer reviewer and study section chair for the Susan G. Komen for the Cure Foundation and the Department of Defense Ovarian Cancer and Breast Cancer Research Programs.

In summary, in a career spanning more than 25 years, I have devoted my efforts to understanding factors that affect risk for ovarian cancer, breast cancer and menopause. I have conducted original research, giving me a deep appreciation of the advantages and disadvantages of different study designs and the challenges of collecting high-quality data for making etiologic inferences. I also have conducted research involving synthesis of the published literature, with the goal of informing decisions based on the best available evidence. A large proportion of my publications have focused on the epidemiology of ovarian cancer, and many of the others focused on breast cancer or menopause have relevance to ovarian cancer because of shared risk factors for the conditions. Based on my education, experience, and expertise, I

am highly qualified to assess the literature on the use of talc in relation to ovarian cancer and provide an expert opinion to a reasonable degree of medical certainty.

Purpose

The purpose of this report is to summarize the epidemiologic evidence related to talc use and ovarian cancer risk and to make a judgment as to whether there is sufficient evidence, based on the totality of evidence from epidemiologic investigations as well as laboratory and mechanistic studies, to conclude with a reasonable degree of scientific certainty that talcum powder use Is a causal factor for ovarian cancer.

Throughout the report, the term "talc" will be used to refer to talcum powder products, recognizing that commercial talc products can contain asbestos, talc containing asbestiform fibers (e.g., talc occurring in a fibrous habit), heavy metals such as nickel, chromium and cobalt and fragrances.

Role and Importance of Epidemiologic Studies

It is important to bear in mind that epidemiologic research on factors that are thought to increase risk for cancer in human populations will consist of observational rather than experimental studies. As with most other now-known carcinogens, including cigarette smoke, it is both ethically wrong and pragmatically impossible to conduct randomized controlled trials to investigate whether a given exposure increases risk for cancer in humans. The judgment as to whether talc causes ovarian cancer will be based on epidemiologic studies in which the investigators collected and analyzed information on exposures (i.e., talc use and other risk factors) that the study participants chose to use, rather than studies in which exposures were randomly assigned to the study subjects in an experimental setting.

Observational study designs used in the study of talc and ovarian cancer include cohort and case-control studies, both of which are well-established and generally accepted methods for studying cancer etiology. In a prospective cohort study, a large group of individuals (the cohort) is identified and exposure to various factors hypothesized to affect risk of disease is

assessed at the time of enrollment (baseline). The cohort is followed over time and the analyses focus on whether the exposed group is more or less likely to develop the outcome of interest than the unexposed group. Some of the prominent advantages of cohort studies are that multiple outcomes/diseases can be assessed within the cohort and exposure assessment precedes the development of the disease, limiting recall bias. However, a primary disadvantage of cohort studies, particularly in relation to cancer etiology studies, is that they must enroll tens of thousands of subjects and follow them for long periods of time to accrue enough cases to have a well-powered study. In addition, if cohort studies do not update exposure information after the baseline assessment, the exposure of some individuals in the cohort may be misclassified.

Case-control studies identify individuals with the disease of interest and an appropriate control group of individuals without the disease and assess exposures that are thought to increase or decrease the risk of the disease. The investigators then analyze whether cases are more likely than the controls to have a given exposure. Case-control studies focus on a single disease, therefore they typically collect more detailed risk factor information for that disease than cohort studies. A major advantage of case-control studies is that they are a more efficient design for studying diseases that are less common or have a long latency period. Therefore, they are very commonly used for etiologic studies of cancer. A disadvantage of case-control studies is that they collect exposure information for the cases after they have already been diagnosed with the disease, which raises concerns that cases may recall exposures differently from controls.

Cohort studies and case-control studies each have advantages and disadvantages for assessing talc as a risk factor for ovarian cancer, and one study design is not clearly superior to the other. In addition, specific details related to the conduct of the study such as methods of exposure assessment, length of follow-up and choice of control group can impact the validity of the findings and the interpretation of results. Therefore, rather than making a judgment based only on the overall study design, the evaluation and interpretation of the findings of the studies must consider the strengths and weaknesses of the individual studies. As the results of the

studies are described and evaluated in this report, specific advantages and disadvantages of individual studies will be discussed in more detail.

In contrast to studies on laboratory animals, studies on humans are subject to more variation in exposure assessment and it is impossible to control all other factors that may contribute to disease risk. For these reasons, judgments on causality from epidemiologic research typically are not based on a single study or even a few studies, but are based on the totality of evidence from multiple studies conducted in different study populations, in different locations and across different time periods. Evidence from the epidemiologic investigations is combined with relevant studies from other disciplines, including pathology, animal and mechanistic studies, to make an assessment of the evidence for a causal association between genital exposure to talcum powder and ovarian cancer.

Methodology

The methodology I used to assess the epidemiologic evidence on talc use as a causal risk factor for ovarian cancer involved conducting a literature search on PubMed using the terms "ovarian cancer" and "talc" to identify all relevant original studies, systematic reviews, meta-analyses, editorials and commentaries (search most recently updated on October 29, 2018). The search I did returned 131 articles, all of which were systematically considered and assessed as to their relevance to talc as a risk factor for ovarian cancer. Twenty-nine articles were not directly relevant to the question at hand (mostly addressing talc in the treatment of malignant pleural effusions). Of the remaining 101 articles, 36 were reports of original epidemiologic studies directly addressing genital talc exposure and ovarian cancer or meta-analyses of such studies.[14,24-56] Other articles retrieved included studies of occupational talc exposure,[57-62] other original research articles that were not specifically epidemiologic studies of genital talc and ovarian cancer (e.g., studies of endometrial cancer, pathology studies, animal studies, etc.)[63-80] and reviews, commentaries and letters [60,81-120] I also examined reference lists from key articles to identify any additional relevant studies. In addition, I reviewed relevant studies as well as documents provided during the course of discovery process.

The primary focus of my review is the epidemiologic studies of genital talc exposure and ovarian cancer and the meta-analyses, with supporting information from other types of publications, including animal, pathology and mechanistic studies used as appropriate to address biological mechanisms underlying the association between talc use and ovarian cancer.

As I evaluated the individual epidemiologic studies (case-control and cohort studies) that described the risk for ovarian cancer associated with talc use, I did not weight one design more heavily than the other because there are advantages and disadvantages to each design for evaluating talc as a cause of ovarian cancer. I considered the potential biases of individual studies, both those that supported and those that did not support an association between talc and ovarian cancer, and how those biases may have impacted the findings. As I describe in this report, some biases have the potential to lead to an overestimate of the relative risk (e.g. recall bias in case-control studies) while others could result in an underestimate of the relative risk (e.g. incomplete ascertainment of talc use in the cohort studies).

I also considered the studies that combined data from multiple studies – meta-analyses or pooled analyses from multiple case-control studies. These types of analyses are often considered to be some of the strongest evidence for a causal association between an exposure and disease as they provide an estimate of the relative risk that is more statistically robust than individual studies. Data from meta-analyses are particularly important for evaluating exposure-disease relationships such as talc and ovarian cancer where the relative risks from most individual studies are approximately 1.2 to 1.5.

As is standard in epidemiologic research, my assessment of whether there is a causal association between talc use and ovarian cancer was guided by the aspects of a causal relationship described by Bradford Hill during the 1960's. Sir Austin Bradford Hill's writings on causal inference provide an accepted framework for assessing whether a given exposure is a cause of a specific outcome.[121] The aspects of the associations that Hill described are: Strength, Consistency, Specificity, Temporality, Biological Gradient, Plausibility, Coherence, Experiment and Analogy. As his writings clearly state, these viewpoints or perspectives should be taken into account when assessing causality, but are not to be considered absolute criteria and not all must be checked off to make a conclusion of a causal relationship. Specifically, he states "What

I do not believe is that we can usefully lay down some hard-and-fast rules of evidence that must be obeyed before we accept cause and effect. None of my nine viewpoints can bring indisputable evidence for or against the cause-and-effect hypothesis and none can be required as a *sine qua non*." This list of viewpoints was used to guide my assessment of the scientific literature on talc use and ovarian cancer.

It is important to point out that, in the end of this process, the assessment of whether a substance is or is not a causal risk factor for a given disease or condition involves scientific judgment that is made by considering and weighing the evidence. In any given case, it is not unusual for scientists and epidemiologists to weigh the Hill factors differently in reaching a conclusion on the causal inference in question. For example, scientists for many years debated the evidence that cigarette smoking causes lung cancer or asbestos causes lung disease.

Epidemiologic Studies Reviewed

Since 1982, when the first case-control study describing an increased risk for ovarian cancer associated with talc use was reported by Cramer, et al., [50] more than two dozen additional reports of epidemiologic studies have been published.[13,14,24-36,38-44,46-49,51-55,122,123] In some instances, data from a particular study were included in more than one publication, due either to an additional analysis of data from a cohort study with longer duration of follow-up (e.g., [31,34]) or to analyses that combined data from more than one study (e.g., [14,25]). Included in these publications are seven meta-analyses published between 1992and 2018 that combined overall results from nine to 27 studies [35,51,52,54-56]and a pooled analysis published in 2013 that combined individual level data from eight case-control studies.[14]

Strength and Consistency of the Association

The first two aspects of the causal relationship described by Bradford Hill, strength and consistency of association, are deeply intertwined. While Bradford Hill referenced the assumption that a larger relative risk is more likely to reflect a causal association, Hill also clearly stated that we should not be "too quick to dismiss a cause-and-effect hypothesis merely

on the grounds that the observed association appears to be slight. There are many occasions in medicine when this is in truth so." [121]

Seven meta-analyses of genital talc exposure and ovarian cancer [35,44,51,52,54-56] calculated summary relative risks that were very consistent across the publications, ranging from 1.22 to 1.36, all with 95% confidence intervals excluding 1, indicating that women who reported talc use were at statistically significant increased risk for ovarian cancer. Similarly, the pooled analysis of eight case-control studies reported an overall odds ratio of 1.24 (95% confidence interval (CI) 1.15 – 1.33).[14]

To put this in context, it is useful to compare the epidemiologic data related to the strength of the association between genital talc use and ovarian cancer with some other well-accepted exposure-disease associations that have relative risks of similar magnitude and are generally accepted to be causal associations. Some examples of such associations and the relative risks from these exposures estimated from meta-analyses are:

1. Oral contraceptive use and breast cancer, relative risk 1.08 (95% CI 1.003-1.165) for ever versus never use and relative risk 1.21 (95% CI 1.04-1.41) for current or recent use versus never use[16]
2. Menopausal estrogen use and breast cancer, relative risk 1.20 (95% CI 1.06-1.37) for more than 5 years use versus no use[124]
3. Passive smoking (also referred to as environmental tobacco exposure or secondhand smoke) and lung cancer, relative risk 1.27 (95% CI 1.17-1.37) for ever versus never exposure to a spouse who smoked[125]
4. Residential radon exposure and lung cancer, relative risk 1.29 (95% CI 1.10-1.51) for highest versus lowest exposure[126]
5. Trichloroethylene exposure and kidney cancer, relative risk 1.32 (95% CI 1.17-1.50) for occupational exposure.[127]

Each of these exposure/disease associations is widely accepted as a causal relationship in the scientific community and has been judged to be a causal association by the International Agency for Research on Cancer (IARC). [128-130] [131] The estimates of the relative risks for these associations from meta-analyses or pooled analyses are approximately 1.25,[16,124-126,132,133]

which is in the range of estimates of the relative risk from meta-analyses and pooled analyses for the association between genital talc use and ovarian cancer. Therefore, we have evidence of well-established causal associations in which the magnitude of the relative risk is very similar to what has been reported for genital talc use and ovarian cancer.

It is instructive to compare in more detail the epidemiologic data on passive smoke exposure to that of talc and ovarian cancer. Passive smoke exposure, like talc, is a very common exposure in the population that can only be assessed retrospectively through self-report, therefore it is difficult to determine the precise level of exposure. In a meta-analysis of 55 studies published between 1981 and 2006 that examined the risk for lung cancer in never smoking women with passive smoke exposure from their spouses, Taylor, et al. [125] reported a pooled relative risk of 1.27 (95% CI 1.17-1.37). The relative risks from individual studies ranged from 0.66 to 2.57, with 44 of the 55 (80%) individual studies reporting a relative risk or odds ratio >1. In the individual studies, only 10 of 55 (18%) reported statistically significant associations (2 of 7 cohort studies, 3 of 25 case-control studies with population-based controls and 5 of 23 case-control studies). These data show that among the many epidemiologic studies that assessed passive smoke exposure as a risk factor for lung cancer, not all had statistically significant findings and some even reported relative risks less than one, yet the overall conclusion from the totality of the evidence is that passive smoke exposure is causally associated with lung cancer.

The most recent meta-analysis published in 2018 on talc and ovarian cancer by Pennikilampi et al. reported a pooled relative risk of 1.31 (95% CI 1.24-1.39) with values from individual studies ranging from 0.73 to 3.90.[56] This result is consistent with other meta-analyses performed. Twenty-four of the 26 (92%) studies reported a relative risk or odds ratio >1, and statistically significant associations were reported in 14 of the 26 (54%) studies. This comparison illustrates that as compared to the well-established causal association between passive smoke exposure and lung cancer, the association between talc and ovarian cancer has a pooled relative risk estimate of similar magnitude with a greater proportion of the studies reporting relative risks >1 and a greater proportion reporting statistically significant

associations suggesting the evidence for talc and ovarian cancer is as significant as for passive smoke exposure and lung cancer.

These comparisons also illustrate the importance of meta-analyses in epidemiologic research when considering exposures for which the strength of association is approximately 1.5 or less. Individual studies, especially those with smaller samples sizes, may not detect a statistically significant increased risk. When the increased risks in this range are seen repeatedly, even when individual studies are not statistically significant, meta-analysis allows the data to be aggregated to make a conclusion that is more robust statistically. When combining these studies through meta-analysis, the totality of the data shows that there is indeed a statistically significant link between genital talc use and ovarian cancer. This observation has been quite consistent, with findings replicated in studies conducted by different teams of investigators, in different geographic locations within and outside the United States, in different race/ethnic groups and over a span of several decades.

In conjunction with the strength of the association, it is also critical to consider the prevalence of the exposure in the population when evaluating its public health impact. A risk factor that is less strongly associated with a disease but has a high prevalence in the population can be responsible for more cases of the disease than a risk factor that is more strongly associated with the disease but has a low prevalence in the population. A measure of the contribution of a risk factor to a disease is the population attributable fraction (PAF), which is defined as the proportion by which the incidence rates of the outcome in the population would be reduced if the exposure was eliminated.[134] Wu et al. [26] calculated the PAF for ovarian cancer related to talc exposure in their multi-ethnic case-control study in Los Angeles. The odds ratio for genital talc use was 1.46 (95% CI 1.27 – 1.69) and the prevalence of use was 41% among the cases and 31% among the controls. The PAFs for the different ethnic groups ranged from 12.2 to 15.1%, which is interpreted as the proportion of ovarian cancer cases that theoretically could be prevented if genital talc use in the population could be eliminated and there were no changes in other risk factors. In other words, of the estimated 22,440 cases of ovarian cancer diagnosed in 2017,[135] approximately 3,300 of them could theoretically have been prevented if women had not used genital talc. The PAF calculation demonstrates that even with an

estimated relative risk for genital talc use of less than 1.5, its high prevalence of use means that it contributes to a substantial proportion of the ovarian cancer cases in the population.

The overall associations seen in the talc-ovarian cancer meta-analyses as well as in many of the individual studies are statistically significant, indicating an increase in risk of approximately 25 to 30%. While not as high as other relationships like smoking and lung cancer, these relative risks are in line with other generally accepted causal relationships (e.g., second hand smoke and lung cancer). I consider the strength of the association seen in the talc-ovarian cancer epidemiologic studies, to be an important factor in favor of a causal relationship between talc and ovarian cancer, particularly when considered along with the consistency of the association sees across these studies.

As described above, among the more than two dozen studies that have reported on the association between talc use and ovarian cancer, the vast majority of them reported relative risks or odds ratios greater than one, indicating strong consistency in the direction of the effect. The findings from the multiple studies are summarized in seven meta-analyses published since 1992, including two published in 2017-18, that combined overall results from six to 27 studies assessing genital talc exposure and ovarian cancer [35,51,52,54,55] [56] [44] and in a pooled analysis published in 2013 that combined individual level data from eight case-control studies. [14] Of the 27 studies included in Berge et al.'s 2017 meta-analysis [51], 24 were case-control studies (18 population-based,[13,23,25,29,30,32,33,38,39,41,42,44,45,47,50,123,136,137] 5 hospital based, [36,43,46,49,122] and 1 with both hospital and population controls [48]) and three were prospective cohort studies [24,27,31]. The calculated overall relative risks for all studies combined in these meta-analyses were 1.3 (95% CI 1.1 – 1.6),[44] 1.27 (95% CI 1.09-1.48),[55] 1.36 (95% CI 1.24-1.49),[35] 1.33 (95% CI 1.16-1.45),[54] 1.35 (95% CI 1.26 - 1.46),[52] 1.22 (95% CI 1.13-1.30)[51] and 1.31 (95% CI 1.24-1.39)[56] and 1.24 (95% CI 1.15-1.33) in the pooled analysis of eight case-control studies.[14] The conclusions from these analyses were quite consistent, even with additional data accumulating over time, indicating that women who used talc products as compared to women who reported no talc use were at 22 to 36% increased risk for ovarian cancer.

When considering the consistency from a number of different studies and meta-analysis, an epidemiologist should evaluate potential sources of bias including but not limited

to publication bias, recall bias, selection bias and information bias. I discuss each of these below.

Publication Bias: When considering a body of epidemiologic evidence from multiple studies, several concerns arise about the completeness of the published data and whether there is selective publishing of studies that find significant positive associations. These concerns were addressed by two distinct analyses conducted in the most recent meta-analyses by Berge, et al. (2017) and Penninkilampi and Eslick (2018).[51,56] The first approach reported was a funnel plot, which is a graphical technique that plots the relative risks derived from the studies on one axis and the standard error of the relative risk (an indicator of the size of the study) on the other. The concept driving this approach is that studies should cluster around the “true” relative risk in the population. Due to random statistical variation, some studies will have relative risks that are higher than the true relative risk and some will be lower than the true relative risk. As sample sizes increase, there should be more precise estimates of the relative risk, therefore larger studies would be expected to produce estimates closer to the true relative risk whereas smaller studies may produce results that deviate further from the relative risk in the overall population. When the study results are plotted, one would expect them to fall into a funnel shape, with the larger studies at the point of the funnel, clustered around the true relative risk in the population, and smaller studies, with more variation in results, showing greater deviation from the average, forming the wide part of the funnel. Notably, in these meta-analyses, the two studies with the highest relative risk estimates (Chen, et al [45] with a relative risk of 3.90 and Godard, et al. [38] with a relative risk of 2.49) and the two studies with the lowest relative risks (Hartge, et al. [49] and Gonzalez, et al. [24]) all had a modest number of cases (<=170).

A funnel plot provides a method for assessing publication bias, i.e., the bias that results from studies with statistically significant findings being more likely to be published than studies that show no association. If one is concerned that studies that showed no association between the exposure and outcome are less likely to be published, the funnel plot allows the visual assessment of this potential bias. A lack of symmetry in the funnel plot, with a deficit of studies showing no association between the exposure and outcome, would be an indication of

publication bias. The papers by Berge, et al. [51] and Penninkilampi and Eslick [56] which are the only meta-analyses that specifically addressed publication bias, concluded that there was no serious publication bias based on both visual inspection of the funnel plot and a statistical assessment of the data from the funnel plot. Therefore, there is a high level of confidence that there has not been preferential publication of studies that found a positive association between talc and ovarian cancer.

A second approach used by Berge, et al. [51] was a cumulative meta-analysis, in which they showed the estimated summary relative risks over time from the first published report in 1982 through the most recently published studies in 2016. The plot showed that after the first initial reports, the overall summary estimates stabilized with estimates in the range of 1.2 to 1.25 over the last 25 years even as more and more data accrued from additional published studies.

These quantitative analyses indicate that it is unlikely that there is publication bias in the talc and ovarian cancer literature (i.e., the analyses do not suggest that studies that found talc use to be a risk factor for ovarian cancer were more likely to be published than those that found no association). Furthermore, from a qualitative perspective, it is also unlikely that there is a substantial risk for publication bias. Given the considerable public health interest in the risk for ovarian cancer associated with a widely-used cosmetic product, it is probable that any well-designed and conducted study that addressed this question would be published, even if it had null findings. Notably, one of the most recent studies, the Sister Study,[24] was published even though it found no increased risk for ovarian cancer associated with talc use.

While the overall conclusions from the meta-analysis and pooled analyses are quite consistent, with an overall statistically significant estimate of the relative risk in the range of approximately 1.2 to 1.3, it is important to consider possible reasons for heterogeneity of the estimates between individual studies.

Among the individual studies that have examined the association between talc use and ovarian cancer, the majority have been case-control studies, with only three prospective cohort studies addressing this research question. The meta-analysis by Berge, et al.[51] noted that the summary relative risk was driven by the stronger associations observed for case-control studies

(relative risk = 1.26 (95%CI 1.17 – 1.35) than for cohort studies (relative risk = 1.02 (95% CI 0.85 – 1.20), which leads one to try to understand possible reasons for the differences by study design and to consider the relative advantages and disadvantages of the different study designs, specifically in relation to the study of talc and ovarian cancer. While the cohort studies do not show a statistically significant association for ever use of talc and ovarian cancer overall, the recent meta-analysis by Penninkilampi and Eslick[56] reported a statistically significant association with the invasive serous subtype of ovarian cancer, which is both the most common subtype and the one with the worst prognosis.

Case-Control Studies – Strengths and Weaknesses: Case-control studies, which are very commonly used in cancer epidemiology, have particular advantages for studying a relatively uncommon cancer like ovarian cancer, which has an annual incidence (number of new cases) in the United States of approximately 11 cases per 100,000 women.[138] In this study design, women with ovarian cancer (the case group) are identified by the research team, typically through a cancer registry, shortly after receiving their diagnosis. A control group of women who do not have the disease are also identified and recruited for the study. Both the cases and the controls provide information on their past exposure history. In a typical case-control study, the study participants complete an extensive questionnaire focusing on a broad range of exposures that are hypothesized to either increase or decrease the risk for cancer. In regard to ovarian cancer, a typical questionnaire will include questions on demographic characteristics, reproductive characteristics like pregnancy and contraception, medical characteristics, family history of cancer and lifestyle characteristics such as dietary factors, smoking history, physical activity and talc use. Notably, some of the factors queried about are expected to increase risk (e.g., family history of ovarian or breast cancer, estrogen use during menopause, talc), whereas others are associated with reduced risk (e.g., oral contraceptive use, pregnancies).

One major advantage of a case-control study is that it is possible to identify and recruit a large number of cases within a relatively short timeframe. To illustrate this point, I will use the example of AACES, the case-control study that my colleagues and I initiated in 2010 to study ovarian cancer in African American women and which was the source of the data we used for our 2016 paper on talc and ovarian cancer.[1,13] We have enrolled more than 600 women with

ovarian cancer and more than 700 control women over a period of approximately 6 years, making it by far the largest study of ovarian cancer in African American women. When the grant application was originally submitted to the National Cancer Institute, one reviewer expressed the opinion that a cohort study would be preferable to the case-control design we proposed. In our response to the review, we pointed out that a prospective cohort study was not feasible for studying ovarian cancer in this population if we hoped to obtain meaningful information in a reasonable timeframe. The Black Women's Health Study, a large prospective cohort study, enrolled approximately 60,000 women starting in 1995 with the goal of studying a wide variety of health outcomes in this population.(https://www.bu.edu/bwhs/) In regard to ovarian cancer, after 18 years of follow-up, only 115 cases of ovarian cancer had been diagnosed among women in the cohort.[139] Although a cohort of 60,000 women is a very large epidemiologic cohort, it is still inadequate to study a relatively uncommon disease like ovarian cancer in a time-efficient manner. We successfully made the argument to the reviewers that a case-control study was the only feasible way to investigate the etiology of ovarian cancer in a timely manner in the African American population. This example illustrates why it is to be expected that the majority of the epidemiologic studies of ovarian cancer would be case-control studies.

Although case-control studies are commonly used in epidemiologic studies of cancer, there are potential biases associated with this study design, including selection bias and recall bias. In this study design, the investigator must select a control group of individuals without the disease being studied as a comparison group to determine the relative frequency of the exposures in the case group as compared to the control group. The goal of selecting a control group is to identify a group that is representative of the population from which the cases arose. This is often stated in textbooks as if someone in the control group were to develop the disease being studied, s/he would have been selected as a case for the study. There are many possible strategies for identifying and recruiting population-based controls, including the use of town registry books,[25,50], telephone recruitment through random digit dialing [13,25,29], neighborhood recruitment,[30] driver's license records [25] and electoral rolls.[123] In hospital-based case-control studies, controls are typically selected from other hospitalized patients, with different studies

applying different criteria for eligible diagnoses among the controls, including other cancer diagnoses or specific non-cancer diagnoses. [36,43,46,49,122]

Among the studies included in the recent meta-analyses, six were hospital-based case-control studies.[36,43,46,48,49,122] The individuals that comprised the control group varied between these studies including patients with non-gynecologic malignancies,[36] patients treated for conditions other than gynecologic or malignant diseases,[122] patients treated for conditions other than those related to reproductive history or oral contraceptive use,[46] patients treated for conditions other than gynecologic, psychiatric, or malignant diseases or pregnancy,[49] both hospital patients and population-based controls [48] and hospital visitors.[43] While the use of hospital controls may be efficient, concerns are often raised as to whether the controls are representative of the population from which the cases arose in terms of the exposures they experienced or their underlying risk for cancer. This is a particular concern with the study by Wong, et al, [36] which is the largest of the hospital-based case-control studies and one that found no association between talc use and ovarian cancer (OR=0.92, 95% CI 0.24-3.62). The control group in this study was "female patients treated for non-gynecologic malignancies during the same period". Standard epidemiologic textbooks (e.g., Rothman, *Modern Epidemiology*[140]) state that controls should be selected from the same source population or study base that gives rise to the cases. It is difficult to make the argument that other cancer patients represent the source population from which the ovarian cancer cases arose, which suggests that this was a poor choice of a control group that could have led to biased findings.

Another of the hospital-based studies, the study by Tzonou et al.[43] which reported a relative risk of 1.05, also had a significant limitation. This study was conducted in Greece, and the overall prevalence of talc use in the study population was 3.5%. Given the small sample size and the low prevalence of exposure, this population was ill-suited to study the relation between talc use and ovarian cancer.

As noted in the meta-analysis by Penninkilampi and Eslick, [56] the hospital-based studies were older (published before 2000) and with the exception of the Wong study [36], all were smaller studies that included fewer than 200 cases. The summary odds ratios from the hospital-based studies was lower but not significantly different than the summary odds ratio from

population-based studies (OR 1.22 versus 1.33, respectively),[56] a result that is not surprising given the important limitations in some of the hospital-based studies.

While there is no ideal method for control selection, arguably population-based control recruitment is more likely to result in a control group that is representative of the population from which cases arose. All of the larger case-control studies that investigated talc use and ovarian cancer (i.e., those with more than 500 cases) were population-based,[13,23,25,29,30,33,42,123,137] which should have minimized selection bias.

Recall Bias: Recall bias is another possible bias in case-control studies. Recall bias is defined as systematic error due to differences in accuracy or completeness of recall of prior events or experiences.[134] It is a concern with case-control studies because information on exposures is obtained through interviews or questionnaires completed after the cases have already been diagnosed with the disease. It is thought that people affected with a disease may have given more thought to possible causes of that disease and have more accurate recall of risk factors than a person serving as a control in the study.

A distinction is made between *recall bias*, which arises from cases recalling exposures differently than controls, and *inaccurate recall* of an exposure that is difficult to remember with precision. Recall bias, which is considered differential misclassification between cases and controls, can result in either an overestimate or underestimate of the true relative risk. Inaccurate recall that occurs to a similar degree in cases and controls is considered non-differential misclassification, and for a dichotomous outcome (e.g., ever vs. never use of talc) will typically result in an underestimate of the true relative risk. An exposure like talc use, especially when assessing use over many years, is clearly one that is subject to a certain amount of inaccurate recall. However, inaccurate recall alone would not result in the consistently increased relative risks observed in the vast majority of the case-control studies of talc use and ovarian cancer.

Therefore, recall bias, which theoretically could result in a biased estimate of the relative risk, must be considered. Situations where recall bias would be considered a particular threat to a study's validity would be: 1) the exposure of interest is one that could be considered sensitive (e.g., illicit drug use, induced abortions), 2) the study hypotheses are known to the

study subjects or interviewers, or 3) there has been considerable media attention focused on an exposure.

In regard to the first situation, genital talc use, while addressing a rather personal topic, would not be considered a particularly sensitive topic. One would not expect that women would be disinclined to report its use out of embarrassment or a desire to report what is perceived to be more socially acceptable as has been reported for exposures like induced abortion.[141]

As to the second point regarding the blinding of the interviewers and the study participants to the study hypotheses, this is standard practice in epidemiologic research. In addition, in the typical case-control study, the investigators are collecting a tremendous amount of questionnaire data to address numerous hypotheses and there is not a particular focus on a single exposure. As an example, the questionnaires from AACES and the North Carolina Ovarian Cancer study each took approximately 1 - 1.5 hours to administer and collected information on a large number of exposures including pregnancy history, contraceptive and hormone use, family history of cancer, medical history, psychosocial factors and lifestyle factors. Data were collected on factors that were expected to be associated with increased risk (e.g., family history of cancer, history of infertility, menopausal hormone use, talc use) as well as those expected to be associated with decreased risk (e.g., oral contraceptive use, pregnancies, physical activity). Given the broad range of hypotheses and the numerous exposures that the cases and controls were queried about and the fact that neither cases nor controls were told in advance of the interview about the specific topics that would be covered, it is unlikely that the women with ovarian cancer would have given more thought to their talc use resulting in substantial systematic over-reporting of talc use among cases. This is supported by studies of other cancers that used empirical data to assess the likely effect of recall bias on relative risk estimates when investigators examined numerous exposures and concluded that recall bias did not consistently lead to increased estimates of the relative risk. [142-144]

Further evidence that recall bias in case-control studies does not inevitably lead to an overestimate of the association between a risk factor and exposure comes from a recent review of meta-analyses of observational studies by Lanza et al.[145] This review analyzed a random

sample of 23 meta-analyses of observational studies addressing different exposure/disease associations published in 2013 and compared findings from case-control studies and cohort studies within individual meta-analyses to determine if conclusions from case-control studies were significantly different from those from cohort studies. The authors concluded that there was no significant difference in effect estimates between the case-control and cohort studies, suggesting that the study design did not have an important impact on the conclusions of the meta-analyses. Although recall bias *theoretically* could lead to an overestimate of the association between a risk factor and disease, the empirical evidence indicates that in practice the effect is small in most situations.

The third situation of the effect of media attention on an exposure deserves consideration as there has been reporting in the lay press in recent years about lawsuits involving talc and ovarian cancer. This concern is not relevant to the vast majority of the studies as virtually all of the data collection in the epidemiologic studies of talc and ovarian cancer occurred prior to such litigation. However one notable exception is AACES,[13] which began enrollment in 2010 and included data collected up through August, 2015. At the recommendation of the reviewer who critiqued the manuscript when it was submitted for publication, our group examined the association between talc and ovarian cancer stratified by the date of enrollment. The odds ratio for genital talc use and ovarian cancer was 1.44 for the overall study population and 1.19 for the participants interviewed before 2014. These data do give some credence to the idea that recall bias could have led to the higher odds ratios when including women interviewed during the time when there was more media attention focused on this exposure, however the fact that the association was attenuated but not eliminated when considering the full study population suggests that the association is not due entirely to recall bias.

Another way to approach the issue of whether recall bias is a likely explanation for the association between talc use and ovarian cancer is to consider whether the association was observed for other gynecologic cancers. The data are admittedly very sparse in this regard, however the only published case-control study of talc use and endometrial cancer reported an odds ratio of 0.88 (95% CI 0.68 – 1.14).[67] A study of ovarian cancer that was conducted by

several of the same investigators as the endometrial cancer study used similar methodology, was conducted in a similar timeframe (early to mid-2000s) in the same geographic region (Australia) and reported a similar prevalence of talc use in the study population. In contrast to their endometrial cancer study in which the investigators observed a non-significant inverse association with talc use, the investigators found a statistically significant increased risk for ovarian cancer associated with talc use (odds ratio=1.17, 95% CI 1.01 – 1.36).[123] While this comparison clearly needs to be interpreted cautiously because there is only a single published case-control study of talc use and endometrial cancer, it does provide evidence to suggest that the association between talc and ovarian cancer observed in most case-control studies is not due simply to recall bias.

Cohort Studies – Strengths and Weaknesses: In contrast to the case-control study, the prospective cohort study design is less susceptible to the selection bias and recall bias described above. Women who develop cancer and the comparison group are from the same population (the cohort) so the bias that could arise from improperly selecting a control group is minimized. Similarly, because the exposure information is collected before the diagnosis of cancer, one would not expect that recall of exposures would differ between the women who went on to develop cancer and those who remained free of cancer.

Despite these advantages, cohort studies do have some important disadvantages in relation to studying cancer etiology. The first is that even with large cohorts, it takes many years for a reasonable number of cancers to develop within the cohort, especially for an uncommon cancer like ovarian cancer. When considering the statistical power of a study to assess the association between an exposure and a disease, the size of the cohort is not the only driver of study power. A more critical consideration is the number of cases that develop within the cohort, which in turn is dependent on the length of follow-up of the larger cohort. Therefore, a large cohort with a relatively short duration of follow-up during which time a small number of cases developed among cohort will have low statistical power. In contrast, the total sample size of a case-control study is likely to be much smaller than a cohort study, but if it has a larger number of cases, it will have greater statistical power than the cohort study.

Among the three cohort studies included in the most recent meta-analysis,[56] the Nurses' Health Study reported 307 cases in a cohort of 78,630 women after approximately 14 years of follow-up;[34,146] the Women's Health Initiative reported 429 cases in a cohort of 61,576 women after a mean of 12.4 years of follow-up[27] and the Sister Study reported 154 cases in a cohort of 41,654 women after a mean of 6.6 years of follow-up.[24] Even with tens of thousands of women in these studies, the number of ovarian cancer cases within each cohort is smaller than the number of ovarian cancer cases in many of the case-control studies. In particular, the number of cases within the Sister Study is smaller than the number of cases in any of the case-control studies published since 1993. As described in a commentary by Narod[81], the lack of a significant overall association between ever use of talc and ovarian cancer in the cohort studies may be due to the fact that the despite the large size of the cohorts, the studies were not adequately powered to detect a relative risk of approximately 1.2.

Another limitation of cohort studies that is of greater relevance to the question of talc use and ovarian cancer is information bias related to exposure assessment. Cohort studies are typically designed to examine many different outcomes that develop within the study population over time. The Nurses' Health Study (http://www.nurseshealthstudy.org/selected-publications) and Women's Health Initiative (https://www.nhlbi.nih.gov/whi/references.htm) have reported on many different outcomes including, but not limited to, multiple types of cancer, cardiovascular diseases, fractures, gastrointestinal conditions and mental health. In contrast, case-control studies focus on a single disease, such as ovarian cancer. Because cohort studies are designed to examine diverse outcomes, the questionnaires must obtain data on risk factors that are relevant to a wider variety of diseases. To keep the questionnaire to a manageable length, a cohort study will typically query about more risk factors but in less detail than a case-control study that is focused on a single disease. This is the case with the talc questions, with the cohort studies collecting less detailed information on talc use, especially in regard to duration and frequency of use, than most of the case-control studies.

It is also worth noting that cohort studies are also subject to recall errors, especially when assessing exposures that began early in life. When the cohort studies assessed talc use, they were asking women to recall their past use of the products up to the point of interview,

similar to how exposure is assessed in the case-control studies. In the Nurses' Health Study, the cohort members were aged 36 to 61 at the time talc use was assessed in 1982, and in the Women's Health Initiative, the mean age at enrollment was 63. Because many women initiate use of talc at a young age, the study participants would have been recalling exposures over several decades, and it stands to reason that there would be some errors in recall. Therefore, in cohort studies as in case-control studies, reported talc use was subject to some degree of inaccurate recall. This likely resulted in non-differential misclassification of the exposure, which usually results in an underestimate of the true relative risk.

Another concern with exposure assessment in cohort studies that is highly relevant to the question of talc use in relation to ovarian cancer is that risk factor information can change over time. If the questionnaire data that were collected when the cohort was assembled do not include a comprehensive exposure history to that time point and are not updated over time, the information may not reflect the complete exposure history of an individual in the time before she was diagnosed with cancer. This could result in some talc users being incorrectly identified as non-users or in incorrect estimates of the duration of exposure.

Incomplete exposure assessment is a potential problem for each of the three cohort studies that have reported on talc use and ovarian cancer, however it is a particular issue for the Sister Study [24] which reported a non-significant inverse association between talc use and ovarian cancer (relative risk of 0.73, 95% CI 0.44 – 1.20). Each of the cohort studies assessed talc use at a single point in time and did not update the information at subsequent follow-up interviews. The Nurses' Health Study collected limited information on talc exposure in 1982, and did not collect additional data on talc use in subsequent questionnaires between 1982 and when the results were described in papers published in 2000 [34] and 2010. [146] Similarly, the Women's Health Initiative collected information on talc exposure when the women were enrolled into the study and did not obtain updated information during the years the cohort was followed. Therefore, any use of talc after that single exposure assessment was not captured, and there would be a certain amount of misclassification of the exposure in both the women who subsequently developed ovarian cancer and those who did not. If the misclassification was non-differential, meaning that the degree of misclassification was similar between the women

who developed ovarian cancer and those who did not, the predicted effect would be a bias towards the null.[140] In other words, non-differential misclassification of talc exposure (as a dichotomous variable) would mean that the observed relative risk was not as strong as it would have been if there had been not misclassification.

The degree of misclassification of exposure in the Sister Study [24] is apparently much greater than in the other cohort studies. Use of talc was assessed through questions about personal care products used only in the 12 months prior to enrollment, including genital talc use in the form of powder or spray applied to a sanitary napkin, underwear, diaphragm, cervical cap or vaginal area. This assessment is essentially a “snapshot” of talc use during a short period of time, capturing neither the cumulative use of talc up to that point nor any subsequent use of talc after the baseline interview. Not surprisingly, the reported prevalence of talc use was quite low in this study. The 14% prevalence reported in the Sister Study was markedly lower than the other two cohort studies (40.2% in the Nurses’ Health Study [34] and 52.6% in the Women’s Health Initiative [27]) as well as in nearly all of the case-control studies. In addition to underestimating the prevalence of talc use in their population, their assessment of talc only during the year prior to enrollment probably did not capture exposure during the most relevant period of the woman’s life. As the authors acknowledged in their paper, if latency (the time between exposure and diagnosis of cancer) is 15 to 20 years, the exposure assessments do not accurately reflect the period of risk. The limitations in the assessment of talc use raise serious questions about the validity of the findings from the Sister Study for this particular exposure. It is impossible to predict the direction or the magnitude of the association between talc use and ovarian cancer if the Sister Study had conducted a more complete assessment of the exposure.

A further limitation of the exposure assessment in the Nurses’ Health Study and Women’s Health Initiative is that neither assessed both the frequency and duration of use of talc. This additional limitation has ramifications for assessing dose-response gradients, which will be discussed in a later section of this report.

While cohort studies are often considered a stronger study design for assessing causal relationships between an exposure and outcome, this is not absolutely true for all exposures and outcomes. Rather than making a judgement about the quality of evidence based solely on

study design, it is important to consider study design from a more nuanced perspective and consider whether a cohort or case-control study provides the most optimal assessment of the exposure and outcome. As described above, each of the three cohort studies that has addressed talc use and ovarian cancer risk had substantial limitations in their assessment of talc use within their study population, which weakens their conclusions that talc use is not significantly associated with ovarian cancer risk.

In addition, the Sister Study,[24] which is a study that was designed primarily to examine breast cancer outcomes among women who had a sister with breast cancer, the small number of ovarian cancer cases despite the large size of the cohort and the inadequate assessment of talc exposure arguably make it a much weaker study than some of the larger, well-designed population-based, case-control studies. Notably, this study, with a relative risk estimate of 0.73 (95% CI 0.44 – 1.20) [24] could be considered an outlier as it is only one of two studies that reported a relative risk substantially less than 1, the other being Hartge's 1983 hospital-based case-control study.[49]

Uncontrolled Confounding in Observational Studies: Uncontrolled confounding is a potential concern in both case-control and cohort studies since they are observational studies. If a factor is associated with talc use *and* is a risk factor for ovarian cancer and is not accounted for in the statistical analysis, it could confound the association between talc use and ovarian cancer. In other words, if there is confounding, the increased risk observed with talc use could be due to the failure to account for the other risk factor. Vaginal douching, which was found to be associated with ovarian cancer risk in the Sister Study, was examined as a potential confounder of the association between talc use and ovarian cancer.[24] Their analyses showed that adjusting for douching using statistical modelling had a negligible effect on the association between talc use and ovarian cancer, providing no evidence of confounding. Other studies have either found an association between talc and ovarian cancer when controlling for douching [44] or found no association between douching and ovarian cancer,[49] thus the available data do not support that douching is a confounder of the association between talc and ovarian cancer. Although uncontrolled confounding is a theoretical possibility, to my knowledge, in the more

than 30 years of research on talc and ovarian cancer no such confounder has been identified that could account for the increased risk associated with talc use.

Overall, the meta-analyses indicate a high level of consistency in findings, especially from the case-control studies. Although weaker associations were observed in the cohort studies, the most recent meta-analysis did report statistically significant associations with invasive serous ovarian cancer in the cohort studies as well as in the case-control studies that reported on histologic subtype.[56] As a whole, the weaker associations observed for the cohort studies could be plausibly explained by limited methods used for talc exposure assessment, the limitations described above, including the most recent cohort study by Gonzalez, et al.,[24] which will have the predicted effect of biasing the results towards the null (i.e., showing an effect that is weaker than the true effect).

Taken as a whole, the overwhelming statistical strength of these studies, whose results are replicated over decades across a wide variety of populations and investigators, further supported by consistent meta-analysis, weighs very heavily in favor of a causal inference.

Temporality

Temporality is the only consideration that is an absolute criterion when making a judgment of causality. This criterion states that a cause (the exposure) must precede the effect (the outcome of interest) in time. Both the cohort and case-control studies that examined talc use in relation to ovarian cancer assessed talc exposure that preceded the diagnosis. In cohort studies, the questionnaire data are obtained before any women in the cohort have a diagnosis of ovarian cancer, and in the case-control studies, women with ovarian cancer are asked to report on exposures that occurred before their diagnosis and controls are asked to report on exposures that occurred in a similar time frame. Therefore, there is no question that the exposure assessment captured talc exposure that preceded the diagnosis of ovarian cancer. Nevertheless, this factor is not highly weighted; while its absence would be fatal to a causal inference, its presence is not particularly compelling support for causation.

Biological Gradient

Associations that show evidence of a biological gradient, or dose-response relationship, are considered to have stronger evidence of causality. While the inconsistencies in reported dose-response trends for talc and ovarian cancer have been noted in some meta-analyses and reviews, e.g.,[51,54] there are several considerations about this exposure that should be taken into account.

First, for an association like talc and ovarian cancer, the dose that is most relevant is the amount of talc that actually reaches the fallopian tubes and ovaries. The epidemiologic data rely on measures of external application as a surrogate of the level of exposure, not the actual exposure in the upper genital tract.

Second, there is some inherent inaccuracy in the measurement of the exposure, as the participants in most studies were asked to recall their duration and/or frequency of use over many years.

Third, the dose of talc exposure has been assessed differently across the studies. Some studies assessed only duration of use (months or years), some assessed only frequency of use (e.g., number of days per month) and some used measures of both duration and frequency to come up with a measure of total dose (estimated lifetime number of applications). The limitations of relying on duration or frequency alone as a measure of talc dose are apparent. For certain exposures, oral contraceptive use for example, duration of use is a good measure of total exposure because the pills are taken once daily. In contrast, patterns of talc exposure may be more inconsistent. Some women may use it daily, others only during their menstrual periods, others may apply it only during certain times of the year and others may have still different patterns of use. Measures of exposure based only on duration of use or only on frequency of use could result in inaccurate estimates of total exposure and obscure a dose-response relationship.

Some of the meta-analyses have cited the lack of a clear dose-response relationship as an argument against talc being a cause of ovarian cancer, and when considering measures of either years of talc use or number of applications of talc per month, there is considerable heterogeneity across studies. When considering the studies that examined dose-response associations considering both dose and frequency to estimate the total number of applications

of talc,[13,14,25,29,30,32,35,41], the majority [13,14,25,30,32] did find significant trends of higher risk with more lifetime applications of talc.

Terry, et al. [14] noted in the pooled analysis of eight case-control studies that the trend for increasing risk for non-mucinous ovarian cancers with an increasing number of genital powder applications was significant when non-users were included in the analysis, but the trend was not significant when the analysis was restricted to ever users. The authors therefore concluded that the significant trend was largely due to the comparison of women who had ever used talc versus those who had never used it, suggesting that the dose-response relationship was not a simple linear increase in risk with greater exposure to talc.

While there is evidence of a dose response relationship in the majority of the studies that considered both frequency and duration of use (i.e., total number of applications), these observations are less consistent than the overall association between talc and ovarian cancer. There are several possible reasons why not all studies observed dose-response relationships, even when an overall association was observed in the study. First, there is likely to be greater inaccuracy in the recall of duration of use as compared to ever/never use, which would tend to obscure a dose-response relationship. Second, when “ever-users” were stratified into duration of use categories, it often resulted in strata with small numbers of women, resulting in less stable relative risk estimates within the duration categories. Third, as noted by Terry, et al.[14], the dose-response relationship may not be a simple linear trend. In many of the studies, even the women in the lowest exposed category had hundreds of episode of talc exposure. Because there could have been considerable exposure even among the women in the “low” exposure categories, greater exposure may not have resulted in substantially increased risk and thus a linear trend may not have been apparent.

Overall, biological gradient was given lesser weight in my assessment of the literature, based on: 1) some of the studies that assessed a dose-response relationship evaluated only duration or frequency of use and not total number of applications, 2) duration and frequency of use are subject to more misclassification than ever use of talc, 3) small sample sizes within strata lead to unstable estimates, and 4) there is the possibility of a non-linear dose-response relationship. Nonetheless, even with these limitations, there was still evidence of a dose-

response relationship in the majority of studies that evaluated it based on the total number of applications.

Biologic Plausibility

Biological plausibility refers to whether there is a reasonable biological mechanism through which the exposure could lead to the disease. Hill is quick to point out that biological plausibility depends on the current state of scientific knowledge. Specifically, Hill wrote "It will be helpful if the causation we suspect is biologically plausible. But this is a feature I am convinced we cannot demand. What is biologically plausible depends upon the biological knowledge of the day." It is clear that from these statements that the consideration of biological plausibility does not require that that there is a *proven* biological mechanism to make a judgment of causality between an exposure and disease. Therefore, for this Hill consideration, a scientist looks for biological evidence that might explain the associations that are observed in the epidemiologic studies. In other words, one has to see whether the observed association "makes sense" biologically. In this case, I have considered both clinical plausibility and biological plausibility. Both of these show that the association seen in the epidemiologic studies "makes sense."

It is probably safe to say that our understanding of the complex biological processes that lead from exposure to disease is incomplete for all cancers. In some instances, the precise biological mechanisms by which an exposure leads to disease remain unclear and in others, some mechanisms are well-established but there is not a complete understanding of why some exposed individuals develop the disease and others do not. An example of the former is alcohol consumption as a cause of breast cancer. While alcohol is considered by IARC to be an established cause of breast cancer, [128] recent publications still describe the association as one in which the exact biological pathways are unclear, even though several possible mechanisms have been hypothesized (i.e., metabolism to acetaldehyde or effects on estrogen levels).[147,148] An example of the latter is smoking and lung cancer. Mechanisms of carcinogenesis from constituents of tobacco smoke have been well-described,[149] however it remains unclear as to why some smokers are more susceptible to developing lung cancer. In short, it is important to

recognize that biological plausibility depends on the current state of knowledge and may evolve over time as new evidence emerges.

When considering the likelihood of talcum powder products causing ovarian cancer, there is robust data that leads to the conclusion that there are biologically plausible mechanisms by which this exposure could lead to ovarian cancer. Specifically, 1) talcum powder products can migrate from the perineum through the genital tract to the ovaries and fallopian tubes, 2) talcum powder products can become imbedded in the ovarian tissue; 3) talcum powder products can induce an inflammatory response, and 4) the inflammatory response can result in increased oxidative stress and expression of cytokines, mutagenesis, and cell proliferation.

Pathology studies have demonstrated that particles may ascend the female genital tract from the vagina to the fallopian tubes and ovaries,[150,151] and talc particles have been identified in ovarian tissue.[71,76,78,79] In fact, the FDA's 2014 response to the Citizen's Petition requesting a cancer warning label on cosmetic talc products states that "the potential for particulates to migrate from the perineum and vagina to the peritoneal cavity is indisputable".[152] Therefore, it is highly plausible that application of talcum powder products to the genital area can result in exposure to the ovaries.

It is also plausible that inhalation of talc products could also be a route of exposure leading to cancer. Studies of asbestos exposure indicate that inhalation of asbestos fibers can result in exposures to the peritoneal tissue, through transport through the lymphatic system and/or blood.[153-155] There is strong evidence that such exposure can result in cancer, most notably mesothelioma. Inhalation of talcum powder products could result in similar peritoneal exposure.

Given the evidence that external application of talcum powder products can reach the ovaries either through upward migration through the genital tract or through inhalation and subsequent transport through the lymphatic system and/or blood, there are plausible biological pathways by which talc could lead to the development of ovarian cancer.

It is well-established through several lines of evidence that talc can cause inflammation. The inflammatory properties of talc are exploited for clinical use in talc pleurodesis, a treatment

for malignant pleural effusions or pneumothorax that involves instillation of talc into the pleural space.(https:www.uptodate.com/contents/talc-pleurodesis) The resultant inflammation and fibrosis result in adhesion of the layers of the pleura, closing the pleural space. The inflammatory properties of talc are also evident in that chronic or acute exposure to talc through inhalation which can result in pulmonary talcosis, a chronic inflammation of the lower respiratory tract.[156,157] Animal studies also confirm that talc causes inflammation, as experiments in rats treated with intra-vaginal or perineal talc showed inflammatory changes in the genital tract.[70] Although neoplastic changes were not observed in this experiment, this could be explained by the small number of animals (n=7) in each group or the duration of the experiment (3 months).

Inflammation has been identified as one of the hallmarks of cancer, with both extrinsic and intrinsic pathways described.[158,159] Talc would be characterized as being involved in an extrinsic pathway, in which an exposure or condition results in chronic, non-resolving inflammatory responses. Chronic inflammation can lead to a cascade of cellular events that could result in damage to DNA, increased cell division and generation of inflammatory mediators.

Recent work by Saed, Fletcher, et al. [160,161] describes the role of oxidative stress in the pathogenesis of ovarian cancer and the effects of talc on the oxidative state of ovarian cancer cell lines. Oxidative stress results when the balance between oxidant and anti-oxidant enzymes and molecules in cells is altered, resulting in an excess of reactive oxygen species or reactive nitrogen species. Oxidative stress, which can result from numerous factors including exposure to carcinogens, infection and chronic inflammation, has been shown to affect the initiation, promotion and progression of several types of cancer. Saed, et al. have reported that talc can generate a pro-oxidant state in both normal ovarian epithelial cells and ovarian cancer cells. Exposure to talc resulted in an increase in mRNA levels of certain pro-oxidant enzymes and a decrease in the mRNA of several anti-oxidant enzymes, suggesting a possible cellular mechanism by which exposure to talc could contribute to the development of ovarian cancer.

There is also evidence in the medical literature that talc products contain additional constituents that are known ovarian carcinogens, particularly asbestos.[162-166]

Asbestos is one of the most established carcinogens in our environment, and is associated with a variety of cancers including mesothelioma, lung, larynx and ovarian.[167,168] IARC has stated that “a causal association between exposure to asbestos and cancer of the ovary was clearly established,” based on strongly positive cohort mortality studies of women with occupational exposure to asbestos as well as studies of women with environmental exposure to asbestos.[169] The Occupational Safety and Health Administration has stated that “there is no safe level of asbestos exposure for any type of asbestos fiber” and that asbestos exposures as short as a few days have resulted in cancer (mesothelioma), indicating that even low levels of exposure may be carcinogenic. (https://www.osha.gov/SLTC/asbestos/)

Although it has been often stated that talc products manufactured after 1976 are asbestos-free, evidence from published scientific reports, [57,162] analyses performed on samples manufactured and packaged at different time points after 1976,[170-173] and internal documents and testimony from the defendants demonstrate that statement is inaccurate.[174,175] There is evidence that products manufactured after 1976 are not asbestos-free. Studies from Longo, et al. show that talc products can contain asbestos and talc containing asbestiform fibers (e.g., talc occurring in a fibrous habit).[170,171] Therefore it is reasonable to conclude that women who regularly used talc products, both before and after 1976, were likely exposed to asbestos and talc containing asbestiform fibers through their use of these products.

Analyses of talcum powder products also demonstrate the presence of other constituents such as chromium and nickel which are well established carcinogens, and cobalt which is considered a possible carcinogen.[169,174] I have also reviewed a report analyzing the 150+ known fragrance ingredients in talcum powder products, many of which have been determined harmful to humans.[176] The presence of these substances provide further evidence that exposure to talc products could result in cancer

It is also plausible that even among women recently diagnosed with ovarian cancer, exposure to the pre-1976 talc products, which are generally understood to have contained asbestos and talc containing asbestiform fibers, increased their risk for ovarian cancer. It is well-established that many cancer risk factors have a long latency, which the National Cancer Institute defines as “the time that passes between being exposed to something that can cause

disease and having symptoms". Numerous examples of cancer risk factors with prolonged latency periods exist. For example, lung cancer typically is not diagnosed among cigarette smokers for several decades after initial exposure [177] and having severe sunburns during childhood is a risk factor for melanoma,[178] which has a median age of diagnosis of 63 years. [135]

It has also been reported that the latency period between exposure to asbestos and mesothelioma (the cancer most strongly linked to asbestos exposure), ranges from 15 to more than 70 years.[179,180] The median latency has been estimated at 22 to 32 years, with longer latency periods estimated for women than for men.[179,180] Thus, it is not unreasonable to conclude that exposure to talc products early in a woman's life could result in ovarian cancer decades later.

Further, other established risk factors for ovarian cancer also demonstrate long latency periods. Oral contraceptive use and history of pregnancy are two of the factors that are most consistently reported in association with ovarian cancer (both of which reduce risk). Although, these are "exposures" that typically occur when women are in their teens, twenties or thirties, the median age of diagnosis of ovarian cancer is 61 years, suggesting that events and exposures from early in a woman's reproductive life can influence her risk for ovarian cancer decades later.

The totality of this evidence indicates that there are plausible biological pathways by which use of talc products could lead to ovarian cancer. There is clear evidence that external applications of these products can result in exposure to the ovaries, through upward migration through the genital tract or inhalation exposure. Once exposed, there are plausible biological mechanisms, by which talc itself or constituents of the talcum powder product could lead to carcinogenic transformation of ovarian cells. This includes credible evidence that talc products contain asbestos fibers, a known ovarian carcinogen, regardless of whether they were manufactured before or after 1976. While it is likely that advances in scientific knowledge may further refine our understanding of how talc exposure can lead to ovarian cancer, our current knowledge is adequate to conclude that there are plausible biological pathways leading from talc exposure to ovarian cancer.

I have considered the biologic plausibility that would support and detract from the hypothesis that talcum powder products can cause ovarian cancer. The more persuasive evidence is that talc can migrate to the ovaries through the genital tract and become imbedded in ovarian tissue. It is also plausible that talc could reach the peritoneal cavity through an inhalation route. Regardless of the route of exposure, it is clear that talcum powder products, including constituents like asbestos and fibrous talc, may cause an inflammatory response and oxidative stress that could lead to cell damage. These biologically plausible mechanisms are a persuasive explanation for the consistent increased risk we have observed in the epidemiologic studies. *Simply put, the observed association "makes sense" biologically.* Along with consistency and strength, I considered this a strong factor favoring a causal inference.

Specificity

As described by Hill,[121], if specificity exists between an exposure characteristic and disease, it provides strong evidence of causality. However, he also stated that "one-to-one relationships are not frequent …multi-causation of disease is generally more likely that single causation". Clearly, ovarian cancer has multiple causes, with talc exposure among many known risk factors. From the standpoint of there being a "one-to-one relationship" between talc and ovarian cancer, there is not a high level of specificity. However, given that talcum powder products are particularly associated with epithelial ovarian cancer, especially serous ovarian cancer, it does support that it is a fairly specific relationship. This aspect was given only modest weight, because talc is one of many possible causes of ovarian cancer.

Coherence

It is recognized that the plausibility depends on the current state of biological knowledge. Knowledge of the biological mechanisms for ovarian carcinogenesis (and virtually any other disease) is incomplete and will continue to evolve as further research continues. Coherence, as described by Hill, means that, even if the knowledge of biology of the disease is not well-defined, the "data should not seriously conflict with the generally known facts of the natural history and biology of the disease".[121] Given the current state of knowledge of ovarian

carcinogenesis, the postulated mechanisms by which talc exposure leads to ovarian cancer do not conflict with the current state of knowledge on ovarian carcinogenesis. This aspect was given considerable weight as it is important that the overall evidence fit together in a coherent manner. Taking into account the plausible pathways by which talc products could reach the target tissue, the expected latency period between exposure and disease, and biological mechanisms that are consistent with our knowledge of carcinogenesis, the data are consistent with the natural history and biology of ovarian cancer.

Experiment

As described above, the epidemiologic data on talc use and ovarian cancer are from observational studies, therefore there are no clear cut experimental data on which a causal assessment can be made. Hill acknowledged that experimental data are often not available for the exposure/disease associations under study, but in some circumstances, experimental or semi-experimental evidence is available.[121] For example, if a preventive action is taken to remove the exposure and the incidence of disease declines, there is strong support for a causal relationship. No such experimental evidence is available for talc use and ovarian cancer.

Analogy

The final viewpoint defined by Hill [121] is analogy, whereby evidence of an association with one risk factor would suggest that a similar risk factor could also plausibly be associated with the disease. Because this viewpoint is rather vague, it is often not incorporated into causal assessments. Nevertheless, while I did not weight it heavily, the similarity between asbestos and asbestiform talc – both of which are widely accepted as causing ovarian cancer – is supportive of this viewpoint.

Conclusion

Epidemiologic evidence linking genital talc use to ovarian cancer has been accruing since 1982.[50] As I evaluated this evidence, I considered the results from individual studies with different designs (case-control and cohort) as well as meta-analyses and a pooled analysis of

multiple case-control studies. In my evaluation of the data, I considered the strengths and weaknesses of individual studies, recognizing that there are advantages and disadvantages of both case-control and cohort studies for evaluating talc as a risk factor for ovarian cancer. I used the Bradford Hill framework as a guide for making my weight of the evidence assessment of whether there is evidence for a causal association between talc use and ovarian cancer.

The epidemiologic evidence I evaluated was derived from more than two dozen studies conducted in many different settings. The vast majority of studies reported relative risks or odds ratios greater than one, indicating that women with ovarian cancer were more likely to have used talc products than women without ovarian cancer. Meta-analyses, which combine findings across multiple studies to come up with an overall estimate of risk that is more statistically robust, have consistently reported that there is a statistically significant increased risk for ovarian cancer among women who reported genital talc use. While meta-analyses have noted that the relative risk estimates from case-control studies have been larger than from cohort studies, limitations in all of the cohort studies could explain the weaker associations observed in these studies. It is also noteworthy that the most recent meta-analysis [56] reported significantly increased risks for invasive serous ovarian cancer, which is the most common subtype as well as the one with the worst prognosis, in both cohort and case-control studies.

The epidemiologic studies that have examined talc use in relation to ovarian cancer risk have been conducted in very diverse populations, both within and outside the United States and in women of different race/ethnicities. The consistency of the findings across populations adds credibility to the findings of increased risk of ovarian cancer among talc users.

The relative risk estimates in most studies and the summary relative risk estimates from the meta-analyses are of a magnitude (~1.25-1.30) that is comparable to other common exposures that are causally related to cancer (e.g., passive smoke exposure and lung cancer, oral contraceptive use and breast cancer, menopausal estrogen use and breast cancer, residential radon exposure and lung cancer). Additional evidence supportive of talc being an ovarian cancer risk factor are biologically plausible mechanisms based on inflammation pathways, oxidative stress and the presence of asbestos, asbestiform talc, and other known

carcinogens in talcum powder products. Evidence of a dose-response relationship exists in many of the studies that considered both duration and frequency of exposure.

Based on the evidence in total, it is my opinion with a reasonable degree of scientific certainty that use of talcum powder products can cause ovarian cancer. I reserve the right to modify or refine my opinions based on additional scientific evidence that may emerge on this topic.

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EXHIBIT A

Duke University Medical Center
Curriculum Vitae

Date Prepared: October 2018

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Primary academic department:	Department of Community and Family Medicine Duke University Medical Center
Present academic rank and title:	Professor with tenure, September 2014
Date and rank of first Duke faculty appointment:	July 1, 2000, Assistant Professor
Medical licensure:	N/A
Date of birth:	December 19, 1957
Place of birth:	Kansas City, Kansas, USA
Citizen of:	United States of America

EDUCATION

	Institution	Year	Degree
High School	Bishop Ward High School Kansas City, KS	1975	Diploma
College	University of Kansas Lawrence, KS	1980	B.S. with distinction, Pharmacy
Graduate School	University of North Carolina – Chapel Hill Chapel Hill, NC	1989	M.S.P.H., Epidemiology
	University of North Carolina – Chapel Hill Chapel Hill, NC	1993	Ph.D., Epidemiology

PROFESSIONAL TRAINING AND ACADEMIC CAREER

Institution	Position/Title	Dates
Shalinsky Drugs, Kansas City, KS	Pharmacist	1980-1981
Community Pharmacy, Wrentham, MA	Pharmacist, Manager	1981-1982
Revco Drugs, Durham/Raleigh, NC	Pharmacist, Manager	1983-1993
Department of Epidemiology University of North Carolina - Chapel Hill	Graduate Research Assistant Teaching Assistant	1987-1993
Burroughs Wellcome Research Triangle Park, NC	Epidemiology Research Associate Summer Intern	1988
Department of Epidemiology Lineberger Comprehensive Cancer Center University of North Carolina - Chapel Hill	Research Assistant Professor	1994-1996
Dept. of Epidemiology and Public Health Yale Comprehensive Cancer Center Yale University School of Medicine New Haven, CT	Associate Research Scientist	1997-2000
Department of Epidemiology University of North Carolina - Chapel Hill	Adjunct Assistant Professor Adjunct Associate Professor	2000-2005 2005-present
Dept. of Community and Family Medicine Duke University Medical Center	Assistant Professor Associate Professor (non-tenured) Associate Professor (tenured) Professor (tenured) Clinical Research Unit Director (formerly Site-Based Research Director)	2000-2004 2004-2008 2008-2014 2014-present 2009-present

PUBLICATIONS

Refereed Publications

1. Aldrich TE, Vann D, **Moorman PG**, Newman B. Rapid reporting of cancer incidence in a population-based study of breast cancer: one constructive use of a central cancer registry. *Breast Cancer Res Treat.* 1995; 35: 61-64.

2. Newman B, **Moorman PG**, Millikan R, Qaqish BF, Geradts J, Aldrich TE, Liu ET. The Carolina Breast Cancer Study: integrating population-based epidemiology and molecular biology. *Breast Cancer Res Treat.* 1995: 51-60.

3. Newman B, Mu H, Butler L, Millikan RC, **Moorman PG**, King M-C. Frequency of breast cancer attributable to BRCA1 in a population-based series of American women. *JAMA.* 1998; 279: 915-21.

4. Millikan RC, Pittman GS, Newman B, Tse C-K J, Rockhill B, Savitz D, **Moorman PG**, Bell DA. Cigarette smoking, N-acetyltransferases 1 (NAT1) and 2 (NAT2) and breast cancer risk. *Cancer Epidemiol Biomarkers Prev*. 1998; 7: 371-8.

5. **Moorman PG**, Hulka BS, Hiatt RA, Krieger N, Newman B, Vogelman JH, Orentreich N. Association between high-density lipoprotein cholesterol and breast cancer varies by menopausal status. *Cancer Epidemiol Biomarkers Prev* 1998; 7: 483-8.

6. Rockhill B, **Moorman PG**, Newman B. Age at menarche, time to regular cycling, and breast cancer. *Cancer Causes Control.* 1998; 9: 447-53.

7. Millikan RC, Pittman GS, Tse C-K J, Duell E, Newman B, Savitz D, **Moorman PG**, Boissy RJ, Bell DA. Catechol-O-Methyltransferase (COMT) and breast cancer risk. *Carcinogenesis.* 1998; 19: 1943-7.

8. Marcus PM, Baird DD, Millikan RC, **Moorman PG**, Qaqish B, Newman B. Adolescent reproductive events and subsequent breast cancer risk. *Am J Public Health.* 1999; 89: 1244-7. (PMCID: PMC1508686)

9. Marcus PM, Newman B, **Moorman PG**, Millikan RC, Baird DD, Sternfeld B, Qaqish B. Physical activity at age 12 and adult breast cancer risk (United States). *Cancer Causes Control.* 1999; 10: 293-302.

10. Furberg H, Newman B, **Moorman PG**, Millikan RC. Lactation and breast cancer risk. *Int J Cancer.* 1999; 28; 396-402.

11. **Moorman PG**, Newman B, Millikan RC, Tse C-K, Sandler DP. Participation rates in a case-control study: the impact of age, race, and race of interviewer. *Ann Epidemiol.* 1999; 9: 188-95.

12. Hall IJ, Newman B, Millikan RC, **Moorman PG**. Body size and breast cancer risk in black and white women: the Carolina Breast Cancer Study. *Am J Epidemiol.* 2000; 151: 754-64.

13. Huang W-Y, Newman B, Millikan RC, Schell MJ, Hulka BS, **Moorman PG**. Hormone-related factors and risk of breast cancer by estrogen receptor and progesterone receptor status. *Am J Epidemiol.* 2000; 151: 703-14.

14. Kinney AY, Millikan RC, Lin YH, **Moorman PG**, Newman B. Lifetime alcohol consumption and breast cancer among black and white women in North Carolina. *Cancer Causes Control*, 2000; 11: 345-57.

15. **Moorman PG**, Kuwabara H, Millikan RC, Newman B. Menopausal hormones and breast cancer in a biracial population. *Am J Public Health*. 2000; 90: 966-70. (PMCID: PMC1446270)

16. Marcus PM, Newman B, Millikan RC, **Moorman PG**, Baird DD, Qaqish B. The associations of adolescent cigarette smoking, alcoholic beverage consumption, environmental tobacco smoke, and ionizing radiation with subsequent breast cancer risk. *Cancer Causes Control*. 2000; 11: 271-8.

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21. Schildkraut JM, Calingaert B, Marchbanks PA, **Moorman PG**, Rodrigues GC. The impact of progestin and estrogen potency in oral contraceptives on ovarian cancer risk. *J Natl Cancer Inst*. 2002; 94: 32-8.

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31. Schildkraut JM, Demark-Wahnefried W, Wenham RW, Grubber J, Jeffreys AS, Grambow SC, Marks J, **Moorman PG**, Hoyo C, Ali S, Walther PJ. IGF1 (CA)19 repeat and IGFBP3 -202 A/C genotypes and the risk of prostate cancer in black and white men. *Cancer Epidemiol Biomarkers Prev*. 2005;14: 403-8

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case-control study with three groups of controls. *Cancer Epidemiol Biomarkers Prev*. 2009; 18:1232-8. (PMCID: PMC2700947)

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risk: an Ovarian Cancer Association Consortium study. *Cancer Epidemiol Biomarkers Prev.* 2010; 19: 245-50. (PMCID: PMC2863004)

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Letters

1. **Moorman PG**. Letter re: Breast cancer risk factors. *Drug Topics*. 2002; 146: 16.

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Book Chapters and Invited Papers

1. **Moorman PG**, Hames CG, Tyroler HA. Socioeconomic status and morbidity and mortality in hypertensive blacks. In Brest AN and Saunders E (eds): *Cardiovascular Clinics: Cardiovascular Diseases in Blacks*. FA Davis Company, Philadelphia, 1991, 179-93.
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4. **Moorman PG**, Terry PD. Dairy products and breast cancer. 2003. United Kingdom Dairy Council. (Invited paper)
5. **Moorman PG**, Berchuck A. Comment on: Hormone replacement therapy does not increase risk for ovarian cancer in women with BRCA mutations. *North American Menopause Society First to Know*. Feb. 15, 2006. www.menopause.org/news.html.
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7. Hulka BS, **Moorman PG**. Breast cancer: hormones and other risk factors. *Maturitas.* 2008; 61: 203-213.
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8. **Moorman PG**. Ovarian failure after pre-menopausal hysterectomy. *European Obstetrics & Gynecology*. 2012; 7: 35-8. (Invited paper)
9. **Moorman PG.** Genetic markers for ovarian cancer risk: are we close to seeing a clinical impact? *Personalized Medicine.* 2012; 9: 565-7. (Invited paper)
10. **Moorman PG.** Should women at high risk for cancer use oral contraceptive pills? *Personalized Medicine*. 2015, 12: 533-5. (Invited paper)

Technical Reports

1. **Moorman PG**, Goldstein K, Coeytaux R, Myers ER, Strauss J, Van Houtven C, Shepherd-Banigan M, Brancu M, Von Isenberg M, Van Noord M, Conklin J, Lallinger K, Schmidt R, McBroom Brooks A, Sanders-Schmidler G. Topic Brief: Nutritional needs of older women. Prepared for Office of Women's Health and Agency for Healthcare Research Quality (AHRQ), 2017.
2. Myers ER, Strauss J, Van Houtven C, Goldstein K, Shepherd-Banigan M, Brancu M, **Moorman PG**, Coeytaux R, Von Isenberg M, Van Noord M, Conklin J, Lallinger K, Schmidt R, McBroom Brooks A, Sanders-Schmidler G. Topic Brief: Maternal Health. Prepared for Office of Women's Health and Agency for Healthcare Research Quality (AHRQ), 2017.
3. Strauss J, Brancu M, Myers ER, Anderson S, Van Houtven C, Goldstein K, Shepherd-Banigan M, **Moorman PG**, Coeytaux R, Von Isenberg M, Van Noord M, Conklin J, Lallinger K, Schmidt R, McBroom Brooks A, Sanders-Schmidler G. Topic Brief: Women's Mental Health. Prepared for Office of Women's Health and Agency for Healthcare Research Quality (AHRQ), 2017.

4. Goldstein K, Coeytaux R, Myers ER, Strauss J, Van Houtven C, Shepherd-Banigan M, Brancu M, **Moorman PG**, Von Isenberg M, Van Noord M, Conklin J, Lallinger K, Schmidt R, McBroom Brooks A, Sanders-Schmidler G. Topic Brief: Girls' Health and Obesity. Prepared for Office of Women's Health and Agency for Healthcare Research Quality (AHRQ), 2017.

5. Shepherd-Banigan M, Van Houtven C, Brancu M, Goldstein K, **Moorman PG**, Strauss J, Coeytaux R, Von Isenberg M, Van Noord M, Conklin J, Lallinger K, Schmidt R, McBroom Brooks A, Myers ER, Sanders-Schmidler G. Topic Brief: Family Caregivers for Older Adults. Prepared for Office of Women's Health and Agency for Healthcare Research Quality (AHRQ), 2017.

Non-authored Publications (acknowledged for contributions)

1. Newman B, Millikan RC, King M-C. Genetic epidemiology of breast and ovarian cancers. *Epidemiol Rev*. 1997; 19: 69-79.

2. Millikan R, Pittman G, Tse C-K, Savitz DA, Newman B, Bell D. Glutathione S-transferases M1, Ti, and P1 and breast cancer. *Cancer Epidemiol Biomarkers Prev*. 2000; 9: 567-73.

3. Krajcik RA, Massardo S, Orentreich N. No association between serum levels of tumor necrosis factor-α (TNF- α) or the soluble receptors sTNFR1 and sTNFR2 and breast cancer risk. *Cancer Epidemiol Biomarkers Prev*. 2003; 12: 945-6.

4. Trivers KF, Stewart SL, Peipins L, Rim SH, White MC. Expanding the public health research agenda for ovarian cancer. *J Womens Health*. 2009; 18: 1299-305.

5. Soubry A, Il'yasova D, Sedjo R, Wang F, Byers T, Rosen C, Yashin A, Ukraintseva S, Haffner S, D'Agostino R Jr. Increase in circulating levels of IGF-1 and IGF-1/IGFBP-3 molar ratio over a decade is associated with colorectal adenomatous polyps. *Int J Cancer*. 2012; 131: 512-7.

Presentations and Published Abstracts (selected)

Moorman PG, Newman B, Butler LM, Ostermeyer EA, Friedman LS, Millikan RC, Liu ET, King MC. Inherited susceptibility at BRCA1 in a population-based sample. Society for Epidemiologic Research, Boston, MA, June 1996

Rockhill B, Newman B, **Moorman P**, Millikan R, Weinberg C. Summary attributable fraction and breast cancer risk factors. Society for Epidemiologic Research, Boston, MA, June 1996.

Furberg H, Newman B, **Moorman P**, Millikan R. Lactation and breast cancer risk. Society for Epidemiologic Research, Edmonton, Alberta, Canada, June 1997.

Marcus PM, Newman B, Millikan RC, **Moorman PG**, Baird DD, Sternfeld B, Qaqish B. The association of adolescent body mass index (BMI) and physical activity with breast cancer risk. Society for Epidemiologic Research, Edmonton, Alberta, Canada, June 1997.

Huang WY, Newman B, Millikan RC, Schell MJ, **Moorman PG**. Hormone-related factors and risk of breast cancer by estrogen receptor and progesterone receptor status. Society for Epidemiologic Research, Chicago, MD, 1998.

Hall IJ, Newman B, Millikan RC, **Moorman PG**. Evaluating body size and breast cancer risk among black women. Society for Epidemiologic Research, Chicago, MD, 1998.

Marcus PM, Newman B, Millikan RC, Baird DD, **Moorman PG,** Qaqish B. Breast cancer epidemiology: the case for adolescent exposures. Society for Epidemiologic Research, Baltimore, MD, 1999.

Moorman PG. Menopausal hormones and risk of breast cancer. Carolina Breast Cancer Study Participant Symposium, Chapel Hill, NC, April 2000

Moorman PG, Jones BA, Millikan RC, Hall IJ, Newman B. Race, anthropometric factors, and stage at diagnosis of breast cancer. Society for Epidemiologic Research, Seattle, WA, June 2000.

Hall IJ, Newman B, Millikan RC, **Moorman PG**. Comparative analysis of breast cancer risk factors among African-American women and white women. Congress of Epidemiology, Toronto, Ontario, Canada, June 2001.

Moorman PG, et al. Nuts and bolts of field studies: things they didn't teach you in school. Congress of Epidemiology, Toronto, Ontario, Canada, June 2001. (Invited talk)

Moorman PG, Calingaert B, Vine M, Halabi S, Berchuck A, Schildkraut JM. Comparison of two methods for calculating lifetime ovulatory cycles. Congress of Epidemiology, Toronto, Ontario, Canada, June 2001.

Plummer P, Jackson S, Konarski J, Mahanna E, Dunmore C, Regan G, Mattingly D, Parker B, Williams S, Andrews C, Vannapppagari V, HallS, Deming S, Hodgson E, **Moorman P**, Newman B, Millikan R. Making epidemiologic studies responsive to the needs of participants and communities: the Carolina Breast Cancer Study experience. Conference on Breast Cancer and Environmental Mutagens, Research Triangle Park, NC, September 2001.

Moorman PG. Population-based study of breast cancer among African-American and White women in North Carolina. North Carolina Central University, Durham, NC, January 2003. (Invited talk)

Moorman PG. Medication use and breast cancer risk. Psychiatry and Behavioral Sciences Grand Rounds, Memorial Sloan-Kettering Cancer Center, New York, NY, March 2003. (Invited talk)

Sansbury L, Millikan R, Schroeder J, **Moorman P**, North K, Sandler R. Use of non-steroidal anti-inflammatory drugs, cyclooxygenase-2 Val411Ala polymorphism and association with colon cancer in a population-based study of African Americans and whites. 3rd Annual AACR International Conference, Seattle, WA, October 2004.

Schildkraut JM, Berchuck A, Murphy S, Marks J, **Moorman P**, Calingaert B, Halabi S. Trinucleotide repeat polymorphisms in the androgen receptor gene and risk of ovarian cancer. 3rd Annual AACR International Conference, Seattle, WA, October 2004.

Moorman PG, Sesay J, Nwosu V, Millikan R. Non-steroidal anti-inflammatory drugs, COX2 polymorphism and breast cancer: a study of gene-environment interactions. Triangle Cancer and Disparities Symposium, North Carolina Central University, Durham, NC, March 2005.

Moorman PG. Racial disparities in breast cancer: problem or opportunity? Johnson C. Smith University, Charlotte, NC, September 2005. (Invited talk)

Trivers K, Gammon M, Abrahamson P, Lund MJ, Kaufman J, **Moorman P**, Cai JW, Porter P, Brinton L, Eley JW. Reproductive factors and breast cancer survival in women less than 55 years of age. 4th Annual Conference on Frontiers in Cancer Prevention Research, Baltimore, MD, November 2005.

Moorman PG. The role of epidemiology in the drug development process. University of Ferrara, Ferrara, Italy, May 2006. (Invited talk)

Moorman PG. Racial disparities in breast cancer: risk factors through survival. Women's Health Research Symposium - Untying the Pink Ribbon: Advances in Breast Cancer. University of Maryland School of Medicine. Baltimore, MD, March 2008. (Invited talk)

Moorman PG, JM Schildkraut JM, ES Iversen ES, ER Myers ER, M Gradison M1, N Warren-White N, F Wang. Weight gain after pre-menopausal hysterectomy. Society for Epidemiologic Research, Chicago, IL, June 2008.

Moorman PG. Non-steroidal anti-inflammatory drugs (NSAIDs) as chemopreventives for cancer: are they ready for prime time? Genetics and Environmental Mutagenesis Society 26th Annual Fall Meeting. Research Triangle Park, NC, October 2008. (Invited talk)

Moorman PG. Introduction to cancer epidemiology. Osher Lifelong Learning Institute lecture series. Chapel Hill, NC, September 2009. (Invited talk)

Moorman PG. Challenges of epidemiologic research in the genomic era: the example of ovarian cancer. Environmental Mutagen Society Annual Meeting, St. Louis, MO, October 2009 (Invited talk)

Moorman PG. Epidemiology in clinical research: beyond randomized controlled trials. Duke University Health System Clinical Education and Professional Development Series, Durham, NC, January 2010. (Invited talk)

Moorman PG. Epidemiology in clinical research: beyond randomized controlled trials. Association of Clinical Research Professionals, Durham, NC, June 2010. (Invited talk)

Moorman PG. The role of epidemiology in understanding disparities in breast cancer. Duke Oncology Symposium – Advances in Surgical and Treatment Options for Breast and Colorectal Cancer. Raleigh, NC, May 2011. (Invited talk)

Østbye T, **Moorman P**. Measurement dilemmas: validity, reliability and messy data. Family Medicine Research Seminar Series, Duke University Medical Center, December 2011.

Moorman P, Østbye T. Research that can tell you something: internal and external validity in study design. Family Medicine Research Seminar Series, Duke University Medical Center, November 2011.

Myers ER, Havrilesky LJ, Gierisch J, **Moorman PG**, Dinan MA, Coeytaux R, Urrutia RP, Lowery WJ, Hasselblad V, McBroom AJ, Sanders GD. Using net benefits and acceptability curves to quantify uncertainty about tradeoffs between harms and benefits of oral contraceptives. Society for Medical Decision Making, Phoenix, AZ, October 2012.

Myers ER, Havrilesky LJ, Gierisch J, **Moorman PG**, Dinan MA, Coeytaux R, Urrutia RP, Lowery WJ, Hasselblad V, McBroom AJ, Sanders GD. Net effects of current patterns of oral contraceptive use on potentially fatal outcomes in the United States. Society for Medical Decision Making, Phoenix, AZ, October 2012.

Stouder A, Melcher B, Morgan PA, **Moorman PG**, Lin L, Stem J. Satisfaction Guaranteed? An Analysis of Lecturer Characteristics Associated with Physician Assistant Student Satisfaction. Physician Assistant Education Association Annual Education Forum, Seattle, WA, November 2012.

Moorman PG. The African American Cancer Epidemiology Study (AACES). Ovarian Cancer in Women of African Ancestry Emerging Consortium Meeting. Bethesda, MD, April 2013.

Moorman PG. Ovarian Cancer: What You Need to Know. Duke Cancer Institute, Cancer Awareness Workshop. Durham, NC, May, August and October 2014.

Moorman PG. Ovarian Cancer in African American Women: The Challenges of Studying a Less Common

Cancer in a Minority Population. Duke Cancer Institute Cancer Control and Population Sciences Seminar Series. Durham, NC, July 2014.

CONSULTANT APPOINTMENTS

National Institutes of Health, Center for Scientific Review

- Epidemiology and Disease Control Subcommittee 2 (EDC-2): Oct. 2000, Feb. 2001
- Special Emphasis Panels: Nov. 2001, Mar. 2002, Nov. 2002, Nov. 2003, July 2004, Nov. 2004, June 2005, Mar. 2006, Nov. 2006, Mar. 2007, July 2007, Nov. 2008, June 2009, July 2009, July 2010, Oct. 2010, Sept. 2011, Dec. 2013, Mar. 2017, Nov. 2017
- Small Grants Program for Cancer Epidemiology: Nov. 2001, Mar. 2003, June 2016, Mar. 2017, June 2017, June 2018, Nov. 2018
- National Cancer Institute, Program Project Review: Jan. 2003, Aug. 2003, Dec. 2003.
- SPORE (Specialized Program of Research Excellence) Review: Breast Cancer Feb. 2005, Ovarian Cancer June 2008, Feb. 2009.

Centers for Disease Control and Prevention. Defining the Public Health Research Agenda for Ovarian Cancer. Invited panel participant. Nov. 2008.

Susan G. Komen for the Cure, Study Section Chair for Post-Doctoral Fellowship in Risk and Prevention, 2010.

Susan G. Komen Breast Cancer Foundation, Study Section Chair for Risk, Prevention and Epidemiology, Member of Programmatic Review Committee, 2005 – 2007.

Susan G. Komen for the Cure/Susan G. Komen Breast Cancer Foundation, Scientific Reviewer, 2003 - 2011.

Department of Defense Breast Cancer Research Program Scientific Peer Review, 1998, 2005, 2007, 2008, 2009, 2013.

Department of Defense Breast Cancer Research Program, Study Section Chair for Training - Epidemiology and Prevention, 2013.

Department of Defense Ovarian Cancer Research Program Scientific Peer Review, 2015, 2016.

Department of Defense Ovarian Cancer Research Program, Study Section Chair for Investigator Initiated Research II, 2018.

National Cancer Institute, Center for Global Health, Data Monitoring Committee for United States – Latin American Cancer Research Network, 2013

CODA, Inc., Research Triangle Park, NC. IRB member, 2004.

National Institute of Environmental Health Sciences Special Emphasis Panel, Technical Evaluation of Support Services for Epidemiology, NIEHS Epidemiology Branch. May 1998.

PROFESSIONAL AWARDS AND SPECIAL RECOGNITIONS

Honorary Physician Assistant, Duke University Medical Center Physician Assistant Program - 2018

Certificate of Appreciation, Duke University Medical Center Physician Assistant Program – 2008

Delta Omega, Public Health Honorary – 1994

The Endocrinologist, Editorial Prize for Volume II – 1993

Research Service Award, National Cancer Institute - 1988-91

Edward E. Smissman Award for Medicinal Chemistry, University of Kansas – 1980

Walter F. Enz Award for Pharmaceutical Chemistry, University of Kansas – 1980

Watkins-Berger Scholarship, University of Kansas - 1975-1980

State of Kansas Scholarship, University of Kansas - 1976-1980

ORGANIZATIONS AND PARTICIPATION

University of North Carolina School of Public Health Alumni Association, Epidemiology Section President, 2001-2002.

Society for Epidemiologic Research

American Pharmaceutical Association

TEACHING RESPONSIBILITIES

Courses Taught

Evidence Based Medicine-I, Duke University, Department of Community and Family Medicine, Physician Assistant program. Primary instructor, 2004-2017.

Evidence Based Medicine-II, Duke University, Department of Community and Family Medicine, Physician Assistant program. Co-Instructor, 2003-2018.

Evidence Based Medicine-I, Duke University, Department of Community and Family Medicine, Physician Assistant program. Lecturer and seminar instructor, 2003.

Epidemiology and Research Methods (PAP 255), Duke University, Department of Community and Family Medicine, Physician Assistant program. Seminar instructor, 2001-2002.

Epidemiology of Cancer (CDE 532B), Yale University, Department of Epidemiology and Public Health. Course director, 1997-2000.

Co-developer of departmental Master's Comprehensive Examination, University of North Carolina-Chapel Hill, Department of Epidemiology, 1995-1996.

Cancer Epidemiology (EPID 233), University of North Carolina-Chapel Hill, Department of Epidemiology, Teaching Assistant, 1992-1993.

Principles of Epidemiology (EPID 160), University of North Carolina-Chapel Hill, Department of Epidemiology, Teaching Assistant, 1990.

Student Mentoring

Helena Furberg, MSPH, University of North Carolina, 1996, Committee Member

Pamela M. Marcus, PhD, University of North Carolina, 1997, Committee Member

Stella Chang, MPH, Yale University, 1997, Committee Member

Mary Riciutti, MPH, Yale University, 1999, Committee Chair

Edward A. Lew, MPH, Yale University, 1999, Committee Member

Shelley Goodstine, MPH, Yale University, 1999, Committee Member

Rupal Desai, MPH, Yale University, 1999, Committee Member

Pei-Yu Lin, MPH, Yale University, 2000, Committee Chair

Lisa Calvocoressi, Ph.D., Yale University, 2003, Dissertation Reader

Rebecca Cleveland, Ph.D., University of North Carolina, 2003, Committee Member

Leah Sansbury, Ph.D., University of North Carolina, 2004, Committee Member

Sumitra Shantakumar White, Ph.D., University of North Carolina, 2006, Committee Member

Katrina Trivers, Ph.D., University of North Carolina, 2006, Committee Member

Amy Dailey, Ph.D., Yale University, 2006, Dissertation Reader

Enid Rivera, M.D., Duke University, 2008, 3rd year Medical Student Preceptor

Alexis Gaines, Duke University, 2013, Master's Committee Member

Chioma Erondu, Duke University, 2013-14, 3rd year Medical Student Preceptor

Tolulope Teniola, Duke University 2016-17, 3rd year Medical Student Preceptor

Tengteng Wang, University of North Carolina, 2018, Committee Member

COMMITTEES AND SERVICE

Standing Committee on Misconduct in Research, Duke University School of Medicine, 2017-present

Senior Faculty Advisory Committee, Office for Research Mentoring, Duke University School of Medicine, 2016-present

Academy of Mentors, Office of Faculty Mentoring, Duke University School of Medicine, 2014-16

Society for Epidemiologic Research. Reviewer for Tyroler and Lilienfeld Prize Papers, 2015

Appointments, Promotions and Tenure Committee, Department of Community and Family Medicine, Duke University Medical Center. Committee Member, 2008-2018

Quality Assurance Sub-Committee for Clinical Research Units, Duke University Medical Center, Committee Chair, 2013-2014

Quality Assurance Sub-Committee for Clinical Research Units (formerly Site-based Research Units), Duke University Medical Center, Committee Member, 2011-2013

Path to Independence Faculty Mentoring Program, Duke University School of Medicine, Peer Reviewer, 2012-2018

Society for Epidemiologic Research. Abstract reviewer for annual meeting, 2011

Cancer Prevention, Detection and Control Research Program, Duke University Medical Center, Cancer Control Pilot Study application reviewer, 2010, 2011

Executive Council, Department of Community and Family Medicine, Duke University Medical Center 2009-present

Education Committee, Department of Community and Family Medicine, Duke University Medical Center, 2009-2017

Faculty Search Committee, Cancer Prevention, Detection and Control Research Program, 2010

Duke Cancer Institute, Editorial Advisory Committee Member, 2010-2011

Duke Comprehensive Cancer Center Annual Meeting, Judge for poster presentations, 2009

Director Search Committee, Cancer Prevention, Detection and Control Research Program, 2009

Partners Allied in Research (PAIR) Pilot Grant application reviewer, Cancer Prevention, Detection and Control Research Program, 2005

Editorial Reviewer

American Journal of Epidemiology
Archives of Gynecology and Obstetrics
Breast Diseases
Cancer
Cancer Causes and Control
Cancer Research
Epidemiology
Gynecologic Oncology
International Journal of Epidemiology
Journal of Community Development
J of the Women's American Medical Assn
Lancet
Nutrition and Cancer
Public Health Nutrition
Women and Health
Annals of Epidemiology
Breast Cancer Research and Treatment
British Medical Journal-Cancer
Cancer Biomarkers
Cancer Epidemiology Biomarkers and Prevention
Clinical Breast Cancer
Ethnicity and Disease
International Journal of Cancer
JAMA
Journal of the National Cancer Institute
Journal of Women's Health
Lancet Oncology
Pharmacogenomics
Trends in Molecular Medicine

CURRENT RESEARCH

Epidemiology of breast and ovarian cancer
Ovarian function after hysterectomy
Racial differences in disease risk and outcomes
Medication use and cancer risk
Etiologic factors for uterine fibroids

EXTERNAL SUPPORT - PAST

Principal Investigator	% effort	Title of Project and Funding Source	Total Costs	Duration
Barbara Hulka	25%	High-Density Lipoprotein Cholesterol and Breast Cancer, National Cancer Institute, R03, Supported dissertation research	$72,234	1992 – 1993

Beth Newman	100%	Carolina Breast Cancer Study, Project 2 SPORE in Breast Cancer, National Cancer Institute, P50.	$1,275,000	1992 – 1995
Beth Newman	50%	Carolina Breast Cancer Study, Project 2 SPORE in Breast Cancer, National Cancer Institute, P50.	$2,511,146	1995 - 1996
Patricia Moorman	50%	Medication Use and Breast Cancer in a Bi-racial Population, National Cancer Institute, R29-FIRST Award.	$498,302	1996 – 2002
Patricia Moorman	0%	Carolina Breast Cancer Study Participant Symposium, North Carolina Division of American Cancer Society Small Grant.	$2500	1997
Patricia Moorman	0%	Carolina Breast Cancer Study Participant Symposium, Susan G. Komen Breast Cancer Foundation Small Grant.	$5000	1997
Joellen Schildkraut	10%	Carolina Georgia Center, Cancer Genetics Network, National Cancer Institute, U-24.	$4,028,129	2002 – 2004
Patricia Moorman	0%	Non-steroidal Anti-Inflammatory Drugs and Breast Cancer: A Study of Gene-Environment Interactions among African-American and White Women, Minority Serving Institution Partnership Grant, Pilot Funds.	$28,040	2003 – 2004
Celette Skinner	5%	Partnerships to Eliminate Disparities in Cancer Outcomes and Research, National Cancer Institute, National Cancer Institute.	$517,743	2002 – 2006
Patricia Moorman	0%	Diversity Supplement to RO1 AG020162 Ovarian Failure Among Hysterectomized Women, National Institute on Aging (Note: Grant was awarded but post-doc accepted another position.)	$169,720	2006
Andrew Berchuck	5%	Biological Basis for Chemoprevention of Ovarian Cancer, Department of Defense.	$824,000	2002 – 2007
Stephen Freedland	5%	Weight Loss and Gain and Cancer Free Survival after Radical Prostatectomy in a Multiethnic Cohort. Prostate Cancer Foundation.	$100,000	2007-2009
Joellen Schildkraut	10%	Genetic Modifiers of BRCA1 and BRCA2, Project 3 SPORE in Breast Cancer. National Cancer Institute.	$1,795,862	2003 – 2009
Joellen Schildkraut	10%	Molecular Epidemiology of Ovarian Cancer. National Cancer Institute.	$3,750,000	2003 – 2010

Patricia Moorman	40%	Ovarian Failure among Hysterectomized Women. National Institute on Aging.	$3,781,480	2003 - 2010
Patricia Moorman (Sub-contract PI)	3%	Cancer Genetics Network. National Cancer Institute.	~$25,000	2010 – 2012
Laura Havrilesky	15%	Oral Contraceptive Use for the Primary Prevention of Ovarian Cancer. Agency for Healthcare Research and Quality	$486,476	2010 - 2012
Jeffrey Marks	5%	Atlantic Breast and Gynecologic Clinical Validation Center. National Cancer Institute	>$1,500,000	2010 - 2013
Cathrine Hoyo	5%	Disparities in Cervical Cancer Precursors and Deregulation of Imprinted Genes National Cancer Institute	~$200,000	2012 – 2013
Emanuel Trabuco (Moorman, Duke PI)	2%	Anti-Müllerian Hormone as a Marker of Ovarian Reserve in Women with and without Hysterectomy. Mayo Clinic Internal Funds	$100,000	2012 – 2013
Evan Myers	10%	Systematic Review of Cancer Screening Literature for Updating American Cancer Society Breast Cancer Screening Guidelines American Cancer Society	~$400,000	2013 - 2014
Joellen Schildkraut	9%	Cancer Education and Career Development Training Grant. National Cancer Institute	~$1,400,000	2009 – 2014
Patricia Moorman (Sub-contract PI)	5%	Rare Cancer Genetics Network National Cancer Institute	~$240,000	2010 – 2016
Joellen Schildkraut (Moorman, co-PI)	20%	Epidemiology of Ovarian Cancer in African American Women National Cancer Institute	~$12,000,000	2010 – 2017
Gillian Sanders	10%	Management of Infertility Evidence-Based Practice Center		2015-2016
Gillian Sanders	10%	Management of Labor Dystocia Evidence-Based Practice Center		2016
Gillian Sanders	15%	Office of Women's Health – Topic Development Evidence-Based Practice Center		2016
Evan Myers	5%	Comparing Management Options for Management: Patient-centered Results for Uterine Fibroids (COMPARE-UF) PCORI	~$19,000,000	2014 – 2018

EXTERNAL SUPPORT - CURRENT

Principal Investigator	% effort	Title of Project and Funding Source	Total Costs	Duration
Joellen Schildkraut (Moorman, sub-contract PI)	13%	Exploring Factors Related to Racial Disparities in Ovarian Cancer Incidence and Survival: The OCWAA (Ovarian Cancer in Women of African Ancestry) Consortium National Cancer Institute	~$450,000	2017 - 2021

PERSONAL INFORMATION

Work address: DUMC Box 2715, 2424 Erwin Road, Suite 602, Durham, NC 27705

Work phone #: (919) 681-4557

E-mail address: patricia.moorman@duke.edu

Home address: 3 Skipwith Court, Durham, NC 27707

Home phone #: (919) 419-9301

Marital status: Married

Spouse's name: Allan R. Moorman, Ph.D.